LETTERS TO THE EDITOR

Mouse-directed computers and ulnar sensory neuropathy

While computers can be affected by "viruses", their users may be prone to pressure neuropathy. We recently examined two patients who presented with an isolated ulnar palmar-cutaneous-branch sensory neuropathy resulting from daily use of a mouse-directed personal computer (PC).

Both males, aged 23 (patient 1) and 34 (patient 2), presented with a two week and one month history, respectively, of paresthesia in the ulnar region of the right hand. Physical examination demonstrated sensory loss in the area of the palmar cutaneous branch of the right ulnar nerve. No weakness or atrophy was observed in these otherwise healthy young men.

Sensory nerve conduction studies of the palmar-cutaneous-branch of the right ulnar nerve revealed a 0·7 and 0·6 ms-longer latency compared with the left, in the first and second patient, respectively.[1] The amplitude of the sensory action potential in the first patient was 12 μV at the affected side compared with 19 μV at the contralateral unaffected nerve. In the second patient these values were 14 μV and 21 μV, respectively. The dorsal cutaneous branch of the right ulnar nerve was normal in both patients, as well as needle electromyography and motor conduction studies of both median and ulnar nerves. Clinical and electrophysiological findings suggested an isolated right ulnar palmar-cutaneous-branch sensory neuropathy. After they abandoned the use of this steering device, recovery occurred progressively. Sensory nerve conduction studies of this sensory branch of the ulnar nerve were within normal limits six months later.

Both patients were using their PC for almost one year and had no predisposing factors for ulnar nerve damage. Their history clearly indicated that the region proximal to the wrist crease of the right arm was intermittently compressed while using the mouse (figure). The palmar sensory branch of the ulnar nerve arises proximal to the wrist crease and supplies sensory innervation to the proximal ulnar aspect of the palm. The deep palmar (motor) branch of the ulnar nerve can be damaged by recurrent pressure.[2-4] However, lesion of the palmar sensory branch is very uncommon, especially when occurring from an occupational nature. This type of neuropathy should be recognised in patients using mouse-directed computers and can simply be prevented by using the keyboard. In addition, this case is not reported to discourage the use of this type of steering device, but to limit its use when any sensory disturbance occurs in the proximal ulnar aspect of the palm.

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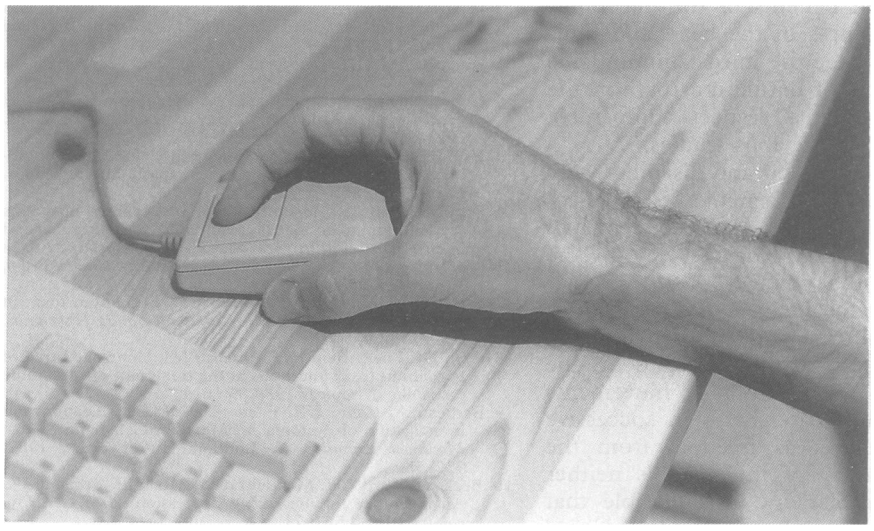

Figure Showing how the region proximal to the wrist crease of the right arm was chronically intermittently compressed while using the mouse.

Oedema associated with the interruption of preganglionic sympathetic tract

Although oedema and swelling of the legs are common in patients with acute transverse myelopathy, the mechanism is poorly understood. We report a patient with multiple sclerosis who showed a marked pitting oedema in the lower limbs following acute transverse myelopathy, which was associated with the interruption of preganglionic sympathetic fibres in the spinal cord.

The patient was a 46 year old female who suffered from the first attack of gait disturbance in April 1989. Sensory impairment below the level of T8, and increased knee and ankle jerks with extensor plantar responses and dysuria were noted. Protein and IgG levels in the CSF were elevated to 71·0 and 17·9 mg/dl, respectively and two oligoclonal IgG bands were detected. No oedema was observed in the legs or feet. Sympathetic skin response (SSR) was studied according to a previously reported method.[1]

The patient lay in a supine position in a warm, quiet room. The skin temperature of the limbs was kept above 31°C. She was asked to open her eyes and not to fall asleep. Standard electromyographic disc electrodes were attached to the palm (G1) and dorsum of the hand (G2) as well as to the sole (G1) and dorsum of the foot (G2) bilaterally. Different types of stimuli were used to evoke the SSR. The endogenous stimulus was deep inspiration. Exogenous stimuli consisted of constant voltage of 50-150 V in single 200μs square pulses, applied to both supraorbital nerves. More than 10 electrical stimuli were administered at irregular intervals greater

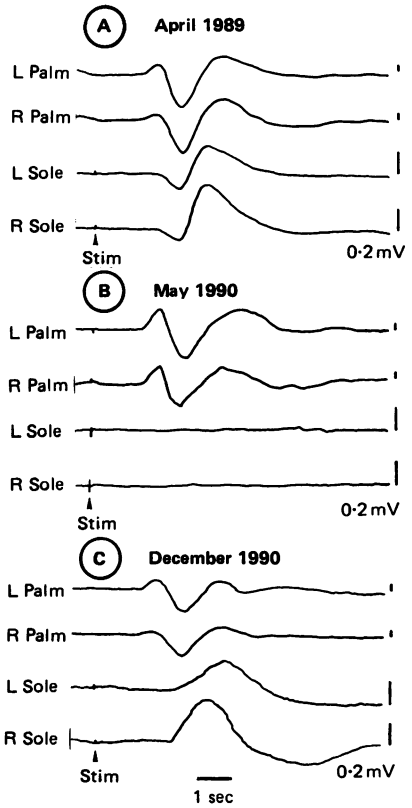

Figure 1 Sympathetic skin response of the patient. The plantar responses disappeared with the appearance of oedema in the legs in May 1990 (B). In December 1990 the oedema in the legs subsided and the plantar responses reappeared (C).

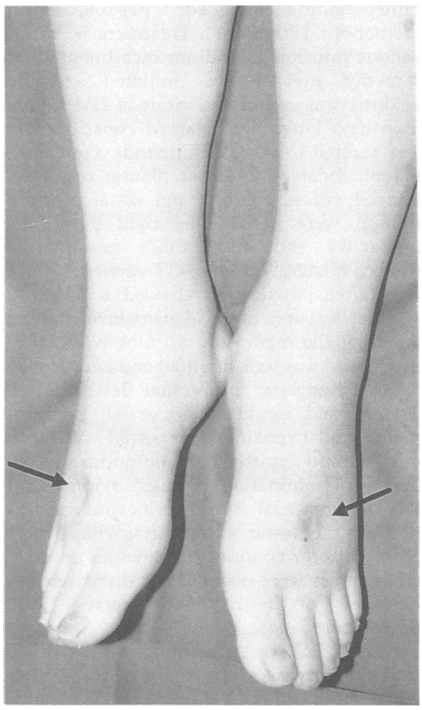

Figure 2 Photograph of the legs, showing marked pitting oedema (arrow).

than 30 seconds apart. The skin responses were normal in the palms and soles (fig 1A). With oral administration of prednisolone the symptoms improved. In March 1990 she had the second attack of transverse myelopathy when she did not have prednisolone. Sensory loss below the level of T5, paraplegia with pyramidal feature, marked pitting oedema of the shins and feet (fig 2), constipation and abdominal distention, and dysuria were observed. Oedema was not noted in the hands or face. The skin temperature in the legs was increased compared with that before the second attack. The oedema was exacerbated and improved in accordance with the abdominal distention and dysuria. There were no signs or abnormal laboratory data suggesting heart failure, renal failure, liver dysfunction, thyroid dysfunction or local inflammation. Venography of the left leg did not show obstruction in the deep veins.

MRI showed lesions with high signal intensity on T_2-weighted image at the lower cervical and the mid-thoracic segments. There were no abnormal results in brain MRI, visual evoked potential, brainstem auditory evoked potential, or somatosensory evoked potential by stimulation of the median nerves. On SSR the plantar responses bilaterally disappeared while the palmer responses were well preserved (fig 1B). Postganglionic function was evaluated with local sweat response to intradermal injection of 10^{-4} g/ml acetylcholine using silastic impression mold technique.[2] The density of sweat droplets at the dorsum of the right foot was normal. After plasma exchange and intravenous administration of prednisolone, the motor impairment and oedema gradually improved. In December 1990 the foot oedema was minimal and the plantar SSR responses had reappeared (fig 1C).

Swelling and oedema is often observed in patients with Raynaud's disease or causalgia after acute interruption of post-ganglionic sympathetic fibres such as a wide-spread sympathectomy. Complete sympathetic block dilates vein and capillary and increases peripheral pooling, which raises hydrostatic pressure within the vessels causing localised oedema. We showed that the preganglionic sympathetic tract in the spinal cord was often disturbed in patients with multiple sclerosis with myelopathy.[1] Most patients with complete transection of the spinal cord due to injury showed swelling of the lower limbs or oedema, but they gradually subsided within several months even without restoration of somatic function. Probably some compensatory mechanism improves the hydrostatic condition in the chronic stage and explains why oedema is not noted in patients with chronic autonomic failure syndrome. On the other hand, SSR was normally recorded in three patients with marked lower limb oedema due to nephrotic syndrome or hypothyroidism. Therefore, it was considered in this patient that the acute thoracic cord lesion interrupted the sympathetic tract, which participated in the production of oedema in the legs. It is suggested that preganglionic sympathetic lesion in the spinal cord should be considered when investigating the cause of oedema in patients with acute myelopathy.

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Incremental response to repetitive stimulation in Guillain-Barré syndrome

The integrity of neuromuscular synaptic transmission may be studied by repetitive stimulation. In a normal individual, the amplitude of the compound muscle action potential (CMAP) changes very little on repetitive stimulation at slow (3 Hertz) or fast (50 Hertz) rates.[1] We recorded an incremental response to repetitive stimulation in a patient with Guillain-Barré syndrome (GBS), who had clinical features resembling those seen in botulism.

A five year old girl was admitted with a cough lasting a week, double vision and difficulty raising her eyelids for four days. On examination she had bilateral lower motor neuron facial weakness, bilateral loss of abduction of the eyes and restriction of upgaze, and a bilateral bulbar palsy. The pupil size and responses were normal. She had moderate proximal weakness of the right upper and lower limbs and all reflexes apart from the right ankle jerk were absent. By the third hospital day she developed complete ophthalmoplegia, complete bilateral ptosis and weakness in all four limbs but distal and left upper limb proximal strength were relatively preserved.

Investigations showed a leukocyte count of 9.6 × 10^9/L. Cerebrospinal fluid on the seventh hospital day contained protein 2·27 g/L, with no cells. A cranial CT scan was normal. A mouse injection test of the patient's serum for botulinum toxin was negative. Stool culture for *Clostridium botulinum* was negative.

Upper limb nerve conduction studies showed the following (table): a) Reduction of resting CMAP amplitude; b) Motor conduction velocity below 80% of the lower limit of

Table Repetitive stimulation and nerve conduction studies

	Repetitive stimulation test			*Nerve conduction study*									
				NCV(ms) [distal latency (m/s)]				*SAP (μV)*				*Fwave (ms)*	
Day No	*CMAP (mV) resting*	*CMAP (mV) 50 Hz**	*Increment (%)*	*RU*	*RM*	*LU*	*LM*	*RU*	*RM*	*LU*	*LM*	*RU*	*RM*
3	**2·6** (L)	4·7	**81**										
7	**2·0** (R)	2·7	37	53 [2·0]	48 [2·5]	50 [**3·7**]	50 [2·6]	17	13	8	15	20·6	22
13	3·6 (R)	3·6	0										
21	3·1 (L)	3·4	9·7	50 [2·0]	46 [3·3]	**44 [3·0]**	**35** [3·0]	**3**	12	**4**	7		**23·8**
28	3·2 (L)			54 [2·2]	**37** [2·6]	54 [2·2]	**NE** [3·1]					**23·8**	**25·0**

*Amplitude of largest CMAP.
(L) = left, (R) = right.
CMAP = compound muscle action potential amplitude.
NCV = forearm motor nerve conduction velocity.
SAP = orthodromic sensory action potential amplitude recorded at wrist.
RU = right ulnar, RM = right median, LU = left ulnar, LM = left median.
NE = no CMAP elicited on stimulation at elbow.
Normal values[3]: Motor conduction velocity: Median = 47–72 m/s; Ulnar = 51–76 m/s.
CMAP amplitude: Median = 2·6–9·7 mV; Ulnar = 3·7–11·6 mV.
Distal motor latency: Median = 1·8–2·8 ms; Ulnar = 1·1–2·2 ms.
Sensory action potential amplitude: Median = 7–36 μV; Ulnar = 7–22 μV.
F wave latency: Median = 16·2–19·8 ms; Ulnar = 15·8–18·8 ms.
Values 20% above or below the limits of normal shown in bold.